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Have we been stretching the truth? The effects of stretching post exercise and delayed onset muscle soreness

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Abstract:

Stretching is a commonly proposed mechanism used to prevent delayed onset muscle soreness (DOMS). Many coaches and medical professionals have always recommended to stretch after exercise to help reduce muscle soreness. Stretching reduces muscle tightness and can help improve flexibility which can help athletic performance. Another accepted belief involving stretching is that it is used to reduce lactic acid levels in the muscle which reduces soreness. There is not sufficient evidence to support either of these claims, however, stretching is commonly used and accepted throughout athletics and active individuals as a way to prevent or reduce muscle soreness. DOMS is muscle soreness that occurs 24-48 hours post-exercise. Eccentric exercise is the main cause to this soreness because it injures the cell membrane of sarcolemma. This injury leads to an acute inflammatory response and results in pain. Lactic acid build-up in the muscle is not the cause of muscle soreness. Pain is specifically the result of eccentric exercise. After reviewing glycolysis and the Cori Cycle, it is understood why lactic acid is not responsible for causing muscle soreness. This review of the literature investigates different studies involving stretching techniques and its effects on muscle soreness. The different types of stretching including ballistic, static, and PNF techniques were compared to each other determine their effects on DOMS and if one technique reduced soreness more than another. The amount of time a stretch was held was also taken into consideration. The literature concluded that stretching after exercise does not reduce muscle soreness caused by DOMS. It did not matter what type of stretching was done or how long the stretch was held. The stretching groups had the same level of soreness as the non-stretching groups. It is still important to stretch to maintain flexibility and reduce muscle tightness, but it has no effects on muscle soreness.

Introduction:

Stretching is a common technique taught to help improve athletic performance. From a young age, it has been taught by parents, physical education, coaches, and medical professionals that stretching is a vital component to exercise. It has been embossed into everyone's mind that stretching will not only improve performance, but it will help reduce muscle soreness. Additional reasons stretching is suggested is that it used to increase range of motion and decreases the risk of injury. It is also widely accepted that stretching post-exercise will decrease the lactic acid levels from the muscle and decrease pain. Coaches have been preaching this theory for many of years, but it is not clear why it was proposed that lactic acid builds up and causes muscle soreness. The main reason lactic acid it is thought to cause pain is because the lactic acid decreases the pH which can lead to injury. There is conflicting research on the true effects of stretching has on delayed onset muscle soreness (DOMS). Many research studies conclude that there is not enough evidence to say people should stop stretching or say people should stretch. In this review of the current literature, the effects that stretching post-exercise has on muscle soreness will be examined as well as what types of stretching are being recommended.

Background:

Stretching is known to cause relaxation in tense muscles by a method of shortening and lengthening the target muscle. It is a technique used to increase range of motion and flexibility in joints, which are the main components that allow movement to occur. Muscle tightness causes a decrease in range of motion and affects an individual's movement. The tightness in the muscle is due to muscle tension which occurs by active tension or passive tension. Passive tension involves the muscle's viscoelasticity and surrounding fascia, whereas active tension involves proprioceptors such as alpha and gamma motor neurons. (1) There are three types of neurons known as sensory, interneurons and motor neurons. Stretching involves the motor (efferent) neurons that are found in the ventral horn and send axons to skeletal muscle. The function of these motor neurons is to trigger a contraction in the target muscle. The two types of motor neurons are alpha and gamma motor neurons. The alpha motor neurons, when activated, release acetylcholine at the neuromuscular junction which leads to a muscle contraction. They are

activated by the interneurons and higher-level motor systems and innervate extrafusal muscle fibers. Gamma motor neurons innervate the muscle spindle. The muscle spindle is a proprioceptive sensory receptor that conveys information about the muscle length and rate of change. Specifically, the Ia sensory fiber within the muscle spindle is responsible for constantly monitoring the velocity of muscle change. The Ia fiber fires when the muscle is stretching and stops firing when muscle length stops changing. When the muscle spindle is contracted or stretched, this stretches the non-contractile portion of the spindle which prompts the sensory endings in the Ia fiber to send information to the spinal cord. This information includes the length of muscle and rate of change in length of the muscle. When the muscle length change stops, the Ia fiber will stop firing and adapt to the new muscle length. The stretch reflex occurs when a muscle spindle detects a fast rate of change in the muscle. The Ia fiber, which innervates the spindle, will then send this sensory information to the interneuron and stimulate the motor neuron of the same muscle to contract. Contracting the muscle will stop over elongation of the muscle and prevent injury. The Golgi tendon organ (GTO) is another proprioceptive sensory receptor that lies connected in the origin/insertion of a muscle/tendon as seen in Figure 1. It relays information about the muscle tendon tension or rate of change of tension in the muscle. It relays this information via the Ib sensory fiber. The Ib sensor fiber innervates the GTO and synapses on the interneuron. The function of the GTO is to sense change in muscle tension. When a muscle generates a force, the sensory endings of the Ib fiber become compressed. This leads to the opening of stretch-sensitive cation channels and causes depolarization generating a nerve impulse to the interneuron. The Golgi tendon reflex prevents the excessive tension in the muscle fibers to prevent injuring the muscle. The reflex occurs when a potentially damaging muscle force is detected by the GTO. The Ib fiber will send this sensory information to the inhibitory interneurons in the spinal cord. The interneurons will then inhibit the activity of the motor

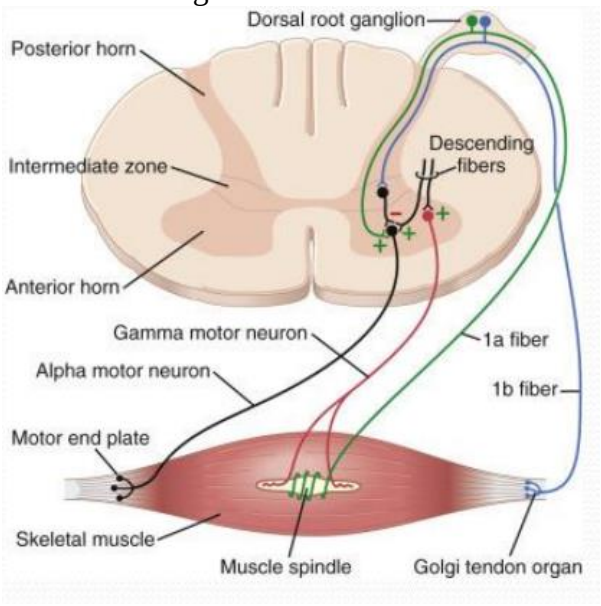

Figure 1 shows the connection between the Ia fiber and muscle spindle and the Ib fiber and GTO. https://o.quizlet.com/WJaCaJfGU3FzOBtTe4VyLw

neurons that control the muscle being contracted. This reduction in motor neuron activity will reduce the muscle contraction, causing relaxation and preventing a possible injury. (1)

There are many types of stretching such as static, dynamic, ballistic and proprioceptive neuromuscular facilitation (PNF) as seen in Figure 2. Static stretching is the most commonly known type. It requires the person to lengthen the tight muscle until a stretched sensation is felt. This pose is then held from 10-60 seconds and then performed on the other side. Each major muscle group is typically only stretched once. Dynamic stretching involves movement and to get into full range of motion. The muscle is lengthened until stretch sensation is felt, but the position is not held for a long amount of time like static. The other difference between static and dynamic, is that the stretch is repeated multiple times and typically performed while walking. Ballistic stretching is a technique that uses the momentum of the body to force a limb into or past full range of motion. It is commonly seen on the sidelines by the athlete swinging their legs side to side, trying to kick higher each time. No research was found as to why these three stretching techniques were first brought into practice, but they have been widely accepted and used for a long time. Proprioceptive neuromuscular facilitation (PNF) works the agonist/antagonist properties of muscles. This means there is a target muscle, the one wanting to be stretched and an opposing muscle. This is type of stretch is typically performed by a medical professional, coach or partner. The provider will lengthen the target muscle until a stretch sensation is felt. The patient will then contract the opposing muscle, usually by pressing against providers hand, then contract the target muscle, relax and provider will further lengthen the target muscle. This is done two to three times per side and each relax-contract is only held about 5-6 seconds.

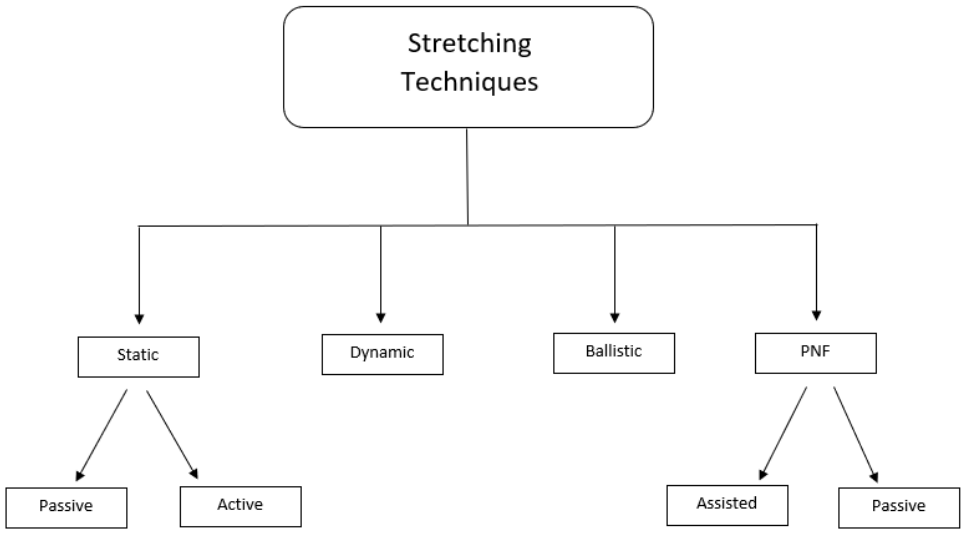

Figure 2 is a map of the different types of stretching.

There are two proposed mechanisms explaining the PNF stretching, as seen in Figure 3. The first mechanism is the autogenic inhibition theory which focuses on the reduction in excitability of a contracted or elongated muscle. (1) This reduction in excitability is a consequence of an increased inhibitory input from the Golgi tendon organ (GTO) in the same muscle. The Ib sensory fiber detects the target muscle being contracted so it sends inhibitory information to the motor neurons causing relaxation in the target muscle. Causing relaxation allows the target muscle to be further stretched. Contracting the target muscle after being lengthened utilizes the autogenic inhibition theory. The second mechanism is reciprocal inhibition. This occurs when the opposing muscle is contracted there is a consequential reduction in activation levels of the target muscle. The motor neurons that activate the opposing muscle, administer an excitatory input to the inhibitory interneurons that relay information to the target muscle. (1) This increase excitatory input to the inhibitory interneurons of the target muscle allows the target muscle to relax and to be further stretched.

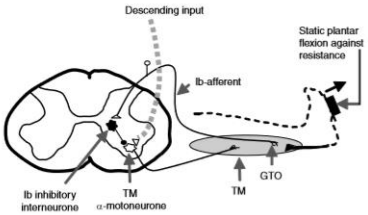

Fig. 1. The mechanism by which autogenic inhibition is purported to contribute to proprioceptive neuromuscular facilitation efficacy. A voluntary static plantar flexion is performed against resistance while the musculotendinous unit (MTU) is on stretch. The plantar flexion developed via descending drive and the existing level of MTU stretch result in an increased firing of tension-sensing mechanoreceptors (Golgi tendon organs [GTOs]) within the same muscle. Increased inhibition from Ib-inhibitory interneurones, a result of the amplified GTO input, results in a reduced level of excitability of the homonymous target muscle (TM), thereby facilitating additional stretch.

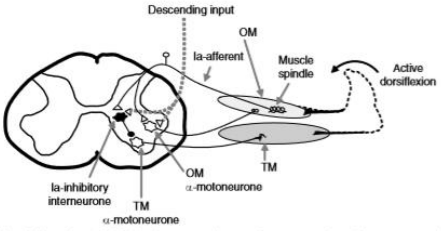

Fig. 2. The mechanism by which reciprocal inhibition is purported to contribute to proprioceptive neuromuscular facilitation efficacy. A shortening contraction of the dorsiflexors (the opposing muscles [OM]) results from descending input onto the OM α-motoneurone. In addition to exciting the OM, descending input and the OM Ia-afferent branch to excite the Ia inhibitory motoneurone. The consequent inhibitory input onto the target muscle (TM) α-motoneurone reduces the activation levels within the same muscle, thereby facilitating additional stretch.

Figure 3 is taken from *Current concepts in muscle stretching for exercise and rehabilitation* and explains the two PNF theories.

Delayed onset muscle soreness is the pain felt in muscles typically 24 to 72 hours after exercising. DOMS is usually the result of an unfamiliar or untrained eccentric exercise. For example, a new exercise or adding more weight or reps to an exercise is a common cause of muscle soreness. Eccentric muscle contraction is the main contributor to muscle soreness. (2) Eccentric contraction is the elongating of the muscle against force. An example of this exercise is

after a bicep curl slowly lowering the weight down. This causes the myofilaments in the muscle fiber, to be contracted and elongated at the same time. In the sarcomere, the myosin cross bridges attach, and the actin starts to move away allowing lengthening. During eccentric exercise, the myosin heads are slowly pulled from the actin sites without using ATP, just power. This forced pulling off of the myosin heads causes damage to the muscle membrane. The soreness and pain felt with DOMS are caused due to injury in the sarcolemma (myofibril sheath) and the sarcomeres of the muscle myofibrils. Sarcomeres are the structural unit of the myofibril. Eccentric exercise causes cell membrane injury leading to an inflammatory response as seen in Figure 4. The acute inflammation response causes the release of prostaglandins and leukotrienes. Prostaglandins causes the sensitization of sensory nerve endings resulting in pain and leukotrienes increase vascular permeability and attract neutrophils. Increased vascular permeability and vasodilation cause swelling which also induces pain. (3)

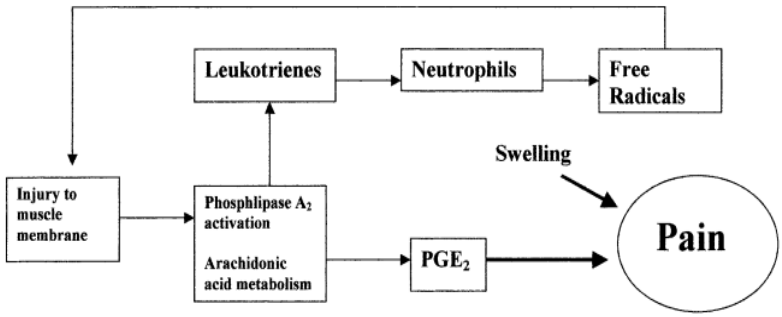

Figure 4 is taken from *Treatment and prevention of delayed onset muscle soreness* (3) and it demonstrates how injury to muscle membrane develops into pain.

It is commonly taught by coaches that delayed onset muscle soreness is due to the buildup of lactic acid, and that athletes need to stretch after exercise to help reduce lactic acid levels to avoid being sore. It is thought the increased lactic acid causes an acidic environment in the muscle which causes pain. (15) This theory is far from the truth. Lactic acid does create an acidic environment in the muscle which is the burning sensation felt while working out. The lactic acid is responsible for causing acute pain, but not the cause of pain from DOMS. Lactic acid does build up in the muscle during exercise, but levels return to normal upon the cessation of exercise. To understand why lactic acid does not build up in the muscle after exercise, there needs to be an understanding of glycolysis. During strenuous exercise, oxygen levels are reduced. This causes anaerobic glycolysis and pyruvate is converted into lactic acid. Glycolysis is a mechanism in which glucose is broken down to get ATP and NADH (energy). Glucose converts glucose to pyruvate through glycolysis. Pyruvate has two options, if it is in the presence oxygen, it will continue to the Krebs Cycle, which is responsible for producing energy by oxidizing Acetyl CoA. If it is in the absence of oxygen or low levels of oxygen, anaerobic

respiration will begin. During anaerobic glycolysis, there is an increase in NADH levels. The elevated levels of NADH stimulates lactate dehydrogenase to deprotonate and pyruvate is converted to lactic acid via lactate dehydrogenase. When oxygen levels are sufficient, the lactate is converted back to pyruvate. This process occurs via the Cori cycle. The Cori cycle begins in the muscle where lactate is produced in anaerobic respiration as seen in Figure 5. When oxygen levels are ample, the lactate will move to the liver. Here it is converted into pyruvate and then glucose via glucogenesis.

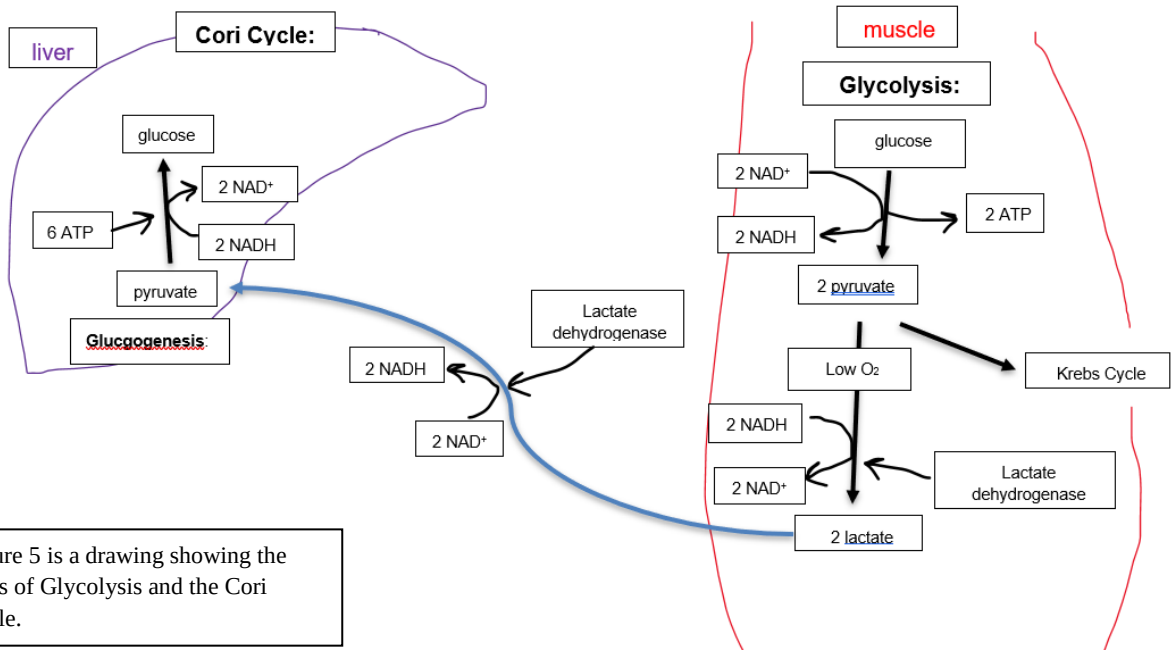

Figure 5 is a drawing showing the steps of Glycolysis and the Cori Cycle.

Blood and muscle lactic acid levels return to homeostasis about 30-60 minutes after recovery. (4) Delayed onset muscle soreness starts displaying symptoms 24 to 72 hours after exercise, which is well after lactate levels have returned to normal. Lactic acid is not the cause of delayed onset muscle soreness. The pain is caused due to the damage of muscle fibers that have been injured due to eccentric exercise.

Review of Literature:

There is a lot of current research done determining the effects of stretching on muscle soreness. Many of the studies look at the different types of stretching and try to determine if one stretching technique is better than the other. Most of the research is carried out using participants

that are young and active. Many of the results concluded stretching doesn't reduce muscle soreness, but there is not enough evidence to suggest that stretching should be stopped. If stretching is not helpful at reducing soreness what should an individual do to help muscle soreness? Should a person still stretch? Is one type of stretching better than the other? How long should a person hold a stretch for? This review of the literature breaks down each of these questions to help determine if we have been stretching the truth.

A randomized trial of stretching before and after activity was done involving 2377 adults. The participants were people whom exercised regularly so they were well trained. The subjects were placed in groups that either stretched before and after activity or did not stretch for 12 weeks. The static stretching group had a decrease in soreness compared to the non-stretch group. However, there was not a significant difference to suggest that stretching reduces muscle soreness. (5) This study has a lot of participants which is great, however the study did not include how pain was measured or how long each static stretch was held. A study was conducted to determine if stretching could induce DOMS. The study looked at the effects of static and ballistic stretching and if DOMS could be caused due to stretching. The participants were all male and randomly assigned to two groups: ballistic stretching or static stretching. Both groups performed three sets of 17 stretches during a 90-minute period. The static group remained stationary and each stretch was held for 60 seconds. Muscle soreness and serum creatine kinase (CK) levels were measured every 24 hours for five days. A 10-point scale was used to measure pain and blood was taken to measure CK levels. Both groups induced significant amounts of DOMS, static producing slightly more soreness. Both groups demonstrated an increase in CK levels. (6) This study helps to understand that CK levels are a good indication of muscle soreness and damage. Creatine kinase is the enzyme that converts creatine phosphate to creatine which produces ATP for the muscle cell. The ATP is produced by the phosphorylation of ADP that comes from creatine phosphate. CK is typically not found in the blood, however, when the sarcolemma is damaged, its contents leak out. The contents include CK and this results in the elevated levels in the blood. This explains why CK levels are a great marker for indicating muscle damage.

A group of physically active and young males from the England professional soccer academy were included in a study. The purpose of this study was to determine the effects of static stretching on muscle soreness. The participants were male and around 16 years old. The

players were broken into to two groups: seated passive stretching for 10 minutes or hold stretch for 15 seconds and repeat twice bilaterally. The players had to participate in three 80-minute games and then performed the stretches after the game. Soreness and creatine kinase (CK) levels were measured before the game, immediately after the game and 48 hours after the match. The study concluded that static stretching did not reduce muscle soreness post exercise because there were elevated levels of CK after 48 hours in the static stretching group. (7) This study is better than others because it uses CK levels as markers of damage. Many of the studies use a pain scale which is subjective data whereas CK levels are objective data. Another strength is that it compares similar stretches to help determine if one type of stretching is possibly better than the other. Unfortunately, neither types of stretching helped reduce muscle soreness.

A study was conducted with 62 male and female volunteers. The purpose of this study was to determine the effects of static stretching and warm up on muscle soreness. There were four different groups that were randomly assigned to the participants. Group A had subjects statically stretch, Group B performed only a step-up warm up, Group C both stretched and did a warm up, and Group D only performed the exercise. The exercise to induce delayed onset muscle soreness was a step test. This test caused participant to do concentric and eccentric work and was performed until exhaustion. Participants then rated their muscle soreness on a scale of 0-6 every 24 hours for 72 hours. The study concluded that neither a warm-up nor static stretching prevented muscle soreness. (8) The strength of this study is that it focused on eccentric exercise to stimulate DOMS. Eccentric exercises are the main way to cause DOMS because it leads to the inflammatory response which causes pain.

McGrath, R. P et al conducted a study to determine the effects of PNF stretching post exercise on DOMS. This study involved young adults as the subjects. It included 57 participants whom volunteered for the study. 19 of the participants were randomized into the PNF group (9 males and 10 females) and 20 participants (14 males and 6 females) were randomized to the static stretch group. 18 participants (5 males and 13 females) were randomized into the no stretch control group. The average age of participants was 18-25 years old. Any participant that was injured was excluded from the study. Participants were advised to not exercise 48 hours prior to the visit so there would be no DOMS present. All groups performed a 5-10-minute jog to warm up then performed a sit and reach test. To induce DOMS, all groups did twelve legs curls three times on each side immediately after the sit and reach test. To ensure muscles were being

eccentrically contracted, the groups were all instructed to lower down their leg to five second count. The static group then performed 10 seconds of static stretching, followed by a 4 second break and another 10 seconds of stretching that same muscle group. The PNF stretching group performed each contract/relax for about 5 seconds each. The no stretching group just sat down for a few minutes. After stretching, all participants performed another sit and reach test. Each participant then reported a pain score 24 hours and 48 hours post-exercise. DOMS was assessed using the Muscle Soreness scale, which is a 6-point scale with different pain descriptions to help the participant judge what pain level they are at on a numerical scale. There was a decrease in DOMS pain from 24 to 48 hours in the PNF and control group, but not the static group. (9) Overall, there was no decrease in pain attributed to DOMS in any of the groups. This study had a good sample size and utilized different stretching techniques to determine the effects they had on DOMS. The limitations to this study include the use of the 6-point pain scale is subjective information and each participant's perceived pain is an individualistic quality. Another issue with stretching, is that the researcher and participant cannot be blinded. There could be bias towards one stretch or the other in the participants because it is commonly thought to reduce soreness. Stretching is also associated with making you feel good which can lead to more bias.

One study was found including female athletes. The purpose of the study was to determine the different effects Kinetic tape (KT), PNF stretching or static stretching had on DOMS. KT is a tape that allows full range of motion where as athletic tape is used to restrict motion. The article proposes the four mechanisms of KT: to increase proprioception, correct muscle function by strengthening weakened muscles, improve circulation of blood and lymph to eliminate tissue fluid and it is thought to decrease pain via neurological suppression. KT has been used for the four proposed mechanisms but lacks sufficient studies to support these effects. The females were randomly split into four different stretching groups. The first group did not stretch but had KT applied to the muscle. The second group performed PNF stretching and the third group did static stretching, holding for 30 seconds and repeating 5 times. The fourth group didn't perform any type of stretch. A pressure algometry was used to measure muscle soreness. (10) A pressure algometry is a good indicator of pain because it measures the pressure pain threshold by applying pressure to a given body part and has a high reliability (r=0.67). (11) The study concluded that none of these therapeutic interventions significantly decreased muscle

soreness. An advantage of this study is the use of the pressure algometry. It is a great tool to measure pain because it has a high reliability and gives the researcher objective data.

Another study that looked at the effects of PNF stretching techniques on the knee flexors. This study involved 24 adults; ages 50-75 years old. There were three different groups that performed various types of PNF stretching. One group did static stretching, one group did contract relax PNF, and the other group did agonist/contract/relax PNF. The measuring tools used to determine the difference in the three groups were range of motion and EMG activity of the muscle. An EMG can be used to objectively show muscle damage. An EMG measures the electrical activity of a muscle. A healthy muscle should show no electrical activity at rest. A damaged muscle will show abnormal activity at rest due to inflammation. (14) The agonist contract relax group had 29-34% more range of motion and 65-100% more EMG activity than others. The PNF contract relax group demonstrated an increase in range of motion but it did not induce muscular relaxation. The strength of this study is that it uses EMG activity to determine muscle damage. Additionally, this study specifically focuses on different types of PNF. There are many different techniques to stretching and this study helps to determine what might possibly be the best way to PNF stretch. Another study looking at the effects of PNF on DOMS included 13 participants that were active adults, 6 males and 7 females. The 13 participants were broken into two groups that either was involved in assisted PNF or unassisted PNF. The assisted PNF group had a provider apply the PNF stretches to the patient. The provider would take patient into a stretch, hold for six seconds, instruct patient to contract stretched muscle by pushing against provider's resistance for 6 seconds. The patient would then relax, and provider would lengthen the muscle more and hold for six seconds. The unassisted PNF group performed this same protocol except used bands instead of a provider and must provide resistance to self. Each contract/relax held for six seconds like the assisted group. The studied concluded that both groups increased range of motion and decreased muscle soreness. (12) PNF stretching is not significantly more beneficial than static stretching post exercise when it comes to preventing muscle soreness, but a few studies suggest there is a trend towards PNF being more beneficial.

An additional study compared PNF and static stretching. The purpose of the study was to compare PNF and static stretching in patients with elbow stiffness. All participants suffered an elbow fracture and then had surgery to fix the fracture. The study was a randomized controlled, single-blind trial. The 40 patients were split into two groups: PNF group or static stretch group

and performed the stretches post-exercise. Both groups underwent the same six-week exercise program. The DASH (Disabilities of arm, shoulder and hand) and visual analogue scale, which is the pain scale using the faces to determine level of pain, were the measuring tools used to determine the pain of the individual. The PNF stretching group demonstrated better scores for the DASH and pain scale compared to static stretching. (13) The strengths of this study are the number of participants and the methodology because it is random and single-blinded. Another strength to this study is the use of the DASH and the fact that it was an upper limb study.

Conclusion:

Static stretching, PNF stretching, dynamic stretching and ballistic stretching demonstrate no reduction or prevention in muscle soreness when trying to treat DOMS. Of all the literature that was reviewed, there are no significant findings to suggest that stretching post exercise eliminates or reduces muscle soreness. Different stretching techniques were examined in the literature and of all the stretching techniques, PNF stretching was the only method that demonstrated a trend in decreasing muscle soreness however the difference is not statistically significant. PNF stretching was the only technique that resulted in an increase in flexibility. DOMS is due to muscle damage and stretching does not physiologically change or reduce the damage to the muscle. Stretching can increase blood flow, which aids in healing however it does not significantly increase the blood flow. The recommended way to reduce muscle soreness is to warm up before exercise by performing a low-level activity for at least 10 minutes. If there is soreness, walking or jogging can help reduce the pain because it will significantly increase the blood flow (4). Increasing blood flow will help to bring oxygen and nutrients to the injured area to aid in healing, and it will flush out metabolic waste from inflammation. From this research, it can be concluded that any type of stretching does not prevent delayed onset muscle soreness.

Discussion:

Stretching is still an important component to wellness because it helps to increase flexibility and therefore having better range of motion to do daily and recreational activities If an individual chooses to stretch, how long should they stretch for? Many of the studies don't include the time a stretching pose is held. Some of the protocols in the reviewed literature

required participants to hold stretch from a range of 20-30 seconds. One study had participants

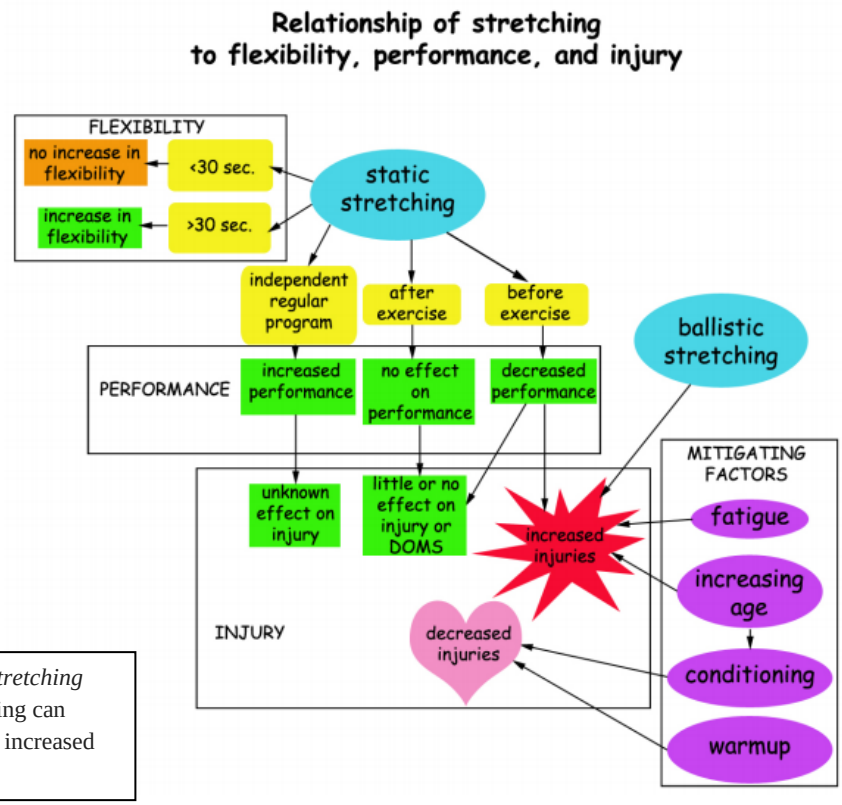

Figure 6 is taken from *The Great Stretching Debate* (4). It shows that conditioning can decrease injury and age and lead to increased injuries.

stretch for 10 seconds, 4 seconds rest, then stretch a little deeper and hold for another 10 seconds. One study instructed participants to hold a stretch for minutes. Mitchell, T., suggests that holding a stretch for 30 seconds is the optimal time as seen in Figure 6. Anything less than 30 seconds doesn't really cause an effect because the muscle will retract to its normal state quickly. If a stretch is held for 30 seconds or more, the muscle will stay in the lengthened state and not as quickly return to normal tension. Stretches held for longer than 30 seconds demonstrated to be no more effective than the 30 second stretch. There are different stretching recommendations for the different age groups. Research concluded that for individuals under 40 years of age should stretch for at least 30 seconds. Anything less than that time showed to be ineffective. For individuals 40 years old and older should hold a stretch for 60 seconds. To get the full effects of stretching, it is suggested to perform the 30 second stretches at least three times a week. Yoga and Pilates are recommended activities to incorporate stretching into your life. (4) Yoga and Pilates are recommended because they focus on balance and core strength not just flexibility. They also work all the major muscle groups to help balance out the body and tone muscles.

When stretching, it is important to remember to not force a stretch, this is when stretching can become harmful.

Further research should be done involving more objective data such as CK levels and EMG activity of muscles. Testing for CK levels in blood and using an EMG, allows the researcher to have subjective proof that damage exists in the muscle. Pain scales aren't the best method to use to measure pain because everyone has their own idea of pain. Pain scales are more of an objective tool to determine the patient's perceived severity of the problem. They are great to use to understand how the patient is feeling, but to determine the severity it is better to use other methods such as blood CK levels or EMG. Another limitation to these studies are that the participants cannot be blinded to the treatment. Participants will know if they are stretching or not. Since stretching a common accepted practice to get rid of muscle soreness, participants could have bias on their results. More research is suggested using older individuals as the subjects and people who are not physically active. Conditioned individuals are less likely to be injured and muscle soreness in conditioned individuals after exercise is typically lower than unconditioned individuals (4). Many of the studies reviewed involved physically active people so it would be interesting to see if there was a difference between the active and inactive. Additional research should be done with an older population because there is not a lot of current research done on that age group. The range of motion, flexibility and muscle strength of older individuals is usually decreased when compared to young and healthy people. The decrease in ROM and muscle strength are due to physiological changes as we age, so a study done with the effects of stretching and muscle soreness with older individuals could be different when comparing to young individuals. Further research should be done involving female subjects. This is a common problem in the research right now. For example, only one study was found using female athletes and 3-4 studies were found with just male subjects. It is important to involve both genders in research to ensure equality and provide the best treatment for everyone.

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